Substitute/replacement pages of the specification are provided in this transmittal.

PLEASE AMEND THE SPECIFICATION AS FOLLOW to incorporate materials previously incorporated by reference: namely,

At page 37, line 7, please correct the paragraph to read as follows: namely,

"The constituents of Formula IV are as set forth (in detail) in Applicant's copending U.S. Patent Application serial No." --09/547,506 (now U.S. 6,548,484-- "incorporated herein by reference in its entirety."

At page 51, line 20, please correct the paragraph to read as follows: namely,

"Additional disclosure of the N-linked glycosyl prodrug pharmaceutical compositions is contained within Applicant's copending U.S. Patent Application Serial No." --09/547,506 (now U.S. 6,548,484--", incorporated herein by reference in its entirety."

At page 51, line 24, please correct the paragraph to read as follows: namely,

"Representative compounds for use according to the instant methods were synthesized as disclosed in co-pending U.S. Patent Application Serial No." --09/547,506 (now U.S. 6,548,484--" incorporated herein by reference in its entirety. Briefly, gluconolactone and 3-hydroxytryamine were reacted slowly in methanol to form a white solid dopamine gluconamide precipitant. The product was collected by filtration, washing and drying *in vacuo* (i.e., dopamine gluconamide, Compound #1, below)."

In the Background Section of the specification at page 3, following the first paragraph, please insert, in order, the following four paragraphs from the Background section of 09/547,506 (U.S. 6,548,484): namely,

> -- In pharmacologic studies conducted over the past 20 years, the results seem to suggest relatively stringent structural requirements for activation of the D1 receptors, particularly in regard to any nitrogen atoms present in the compound (e.g., see Seiler et al., 1991 ;Berger et al., 1989; Brewster et al., 1990; Kaiser et al., 1982; Dandridge et al., 1984; Brewster et al. 1990; Weinstock et al., 1985; Riggs et al. ; Seiler et al., 1982; Shah et al., 1996; Knoerzer et al., 1994). In addition, the nature of the terminal

group (i.e., amino), or presence or length of an n-alkyl chain (Iorio et. al., 1986) may reportedly influence binding interactions at D1 sites. Based on experience with different pharmacophores, several receptor models have been proposed (Seiler and Markstein, 1989; Petersson et. al., 1990; Brewster et. al., 1990; Knoerzer et. al., 1994; Snyder et. al., 1995; Minor et. al., 1994). By comparison, pharmacologic studies of D2-like receptors suggest somewhat less rigid overall structural requirements, but also restrictions around any nitrogen atoms (e.g., see McDermed et al. 1979; Freeman and McDermed, 1982.; Liljefors et al., 1986; van de Waterbeemd et al., 1987). --;

--The Na^+/Cl^- dependent dopamine transporter, DAT1, granule system mediates calcium-dependent outward dopamine release into the synaptic cleft and inward energy-dependent dopamine vesicular re-uptake into the cytoplasm of presynaptic neurons. Loading of biosynthetic dopamine into granules is effected by the vesicular monoamine transporter (VMAT2; reviewed in Miller et. al., 1999). DAT may also control movements of other monoamines in brain tissues. Cocaine, amphetamines, phencyclidine and certain anti-depressants and uptake inhibitors interfere with dopamine transport by DAT (e.g., see Jones et al., 1999; Giros et. al., 1992). DAT function may be regulated by steroid hormones, has second order dependence on Na^+ (Earles et. al., 1999) and may be coupled (or uncoupled) to modulatory second messenger systems, (e.g., down-regulation of DAT accompanying activation of protein kinase C by phorbol esters), and ionic currents (Melikian et al., 1999; reviewed in Figlewicz, 1999). Radiotracer imaging methods have been used to localize DAT (e.g., within the nucleus accumbens and mid-brain regions) and D1 and D2 receptors (e.g., in nigrostrial pathways) in the brains of normal subjects, as well as in patients with Parkinson's disease and neuropsychiatric diseases such as schizophrenia (reviewed in Verhoeff, 1999). Structure activity studies of antagonists have suggested that: (i) the DAT transporter may be sensitive to N-substitution (Choi et al., 2000); (ii) N-phenyl-substituted analogues may inhibit transport (Prakash et al., 1999; Husbands, et al., 1999); (iii) certain energetically unfavored boat conformations of rings may have high affinity for DAT (Prakash et al., 1999); (iv) structural rearrangement of the DAT protein may occur and be required for inward transport (Chen et al., 2000); (v) the DAT protein contains an endogenous Zn^{2+} binding site (Loland et al., 1999); (vi) DAT transporter function is sensitive to aromatic substitutions (Husbands, et al., 1999); and, (vii) apparent ordered kinetics for DAT transporter function is Na^+ binding first, then dopamine and then Cl^-. --;

--Several tissue enzyme systems exist for altering catecholamines, including dopamine. Monoamine oxidases, MAO-A in neural tissues and MAO-B in other tissues including stomach and intestine, are oxioreductases that deaminate dopamine and other catecholamines with preferential activity manifest for 2-phenylethylamine and benzylamine. Catechol-O-methyltransferase is a cytosolic enzyme that catalyzes

addition of a methyl group, usually at the 3 position of a benzyl ring. O-methoxylated derivatives may be further modified by conjugation with glucuronic acid. Non-neuronal dopamine transporter uptake mechanisms may also exist, e.g., in kidney (Sugamori et. al., 1999). --; and,

--Oral delivery of drugs constitutes special chemical challenges, i.e., general simultaneous requirements for intestinal penetration, blood borne delivery, blood-brain-barrier penetrability and maintenance of functional (receptor binding and/or metabolic) utility. CNS active drugs constitute yet additional special and challenging problems, i.e., low pH stability (or protection) and intestinal transport. Intestinal intracellular transport mechanisms for amino acids, vitamins and sugars are varied. Glucose transport has recently been reviewed (Takata et. al., 1997). Transport mechanisms for glucose include intestinal transport vesicles and Na+/glucose co-transporters (SGLTs), i.e., driving active transport of glucose and galactose across the intestinal brush border by harnessing Na^+ gradients across the cell membrane. Net rates of vesicle transport and exocytosis have been estimated to be in the range of 10 thousand to 1 million per second (Wright et. al., 1997). Missense mutations in SGLT1 reportedly result in potentially lethal inability to transport glucose and galactose (Martin et. al., 1996). Certain sugar specificity's, structural requirements and capabilities of Na^+-dependent glucose transport carriers have been investigated with impure receptor membrane preparations, and/or mixtures of receptors, with the findings that the glucosyl transporter in human erythrocytes (i.e., GLUT1): (i) seems to require that the ring oxygen atoms at positions C1, C3, C4, and possibly C6, be capable of forming hydrogen bonds with the transporter protein, and (ii) a hydrophobic group at C5 may increase affinity for the transporter (Barnett et al., 1973). Intestinal glucose transporter mechanisms reportedly prefer: (i) β-anomers to α-anomers; (ii) β-D-glucose to β-D-galactose; and, (iii) β-glucoside > α-glucoside > β-galactoside > α-galactoside. The α-anomers of glucose and galactose were reportedly hydrolyzed to their aglycone constituents during a non-Na^+-dependent desglucosylation transport (Mizuma et. al., 1992, 1993, 1994). Apparently unrelated studies of antiviral glycosides have reportedly found that: (i) C1 phenyl-substituted glycosides and para-substituted butyl-phenyl derivatives may inhibit glucose transporters (Arita et. al, 1980); (ii) C1 O-acyl glycoside derivatives with alkyl chains or carbonyl groups (as an aglycone substituent) may act as non-penetrating inhibitors of glucose transport (Ramaswamy et. al., 1976); and (iii) 1-5-anhydroglucitol and 6-deoxyglucose may be transportable (Alvarado et. al., 1960). Thus, like dopaminergic receptor binding, the art suggests that special chemical structural requirements may exist for intestinal transport. --

At pages 57 to 61 please amend the citations section, as set forth in the pages 57-61A Replacement Sheets, to add the citations to authors incorporated by reference from the "Background" (above) and "Citations" sections of 09/547,506 (U.S. 6,548,484).